# A Post-Graduate Lecture

ON

# TRAUMATIC ENOPHTHALMOS

*Delivered at the West London Post-Graduate College*

BY

H. PERCY DUNN, F.R.C.S. Eng.

SENIOR OPHTHALMIC SURGEON, WEST LONDON HOSPITAL; LECTURER ON OPHTHALMOLOGY AT THE WEST LONDON POST-GRADUATE COLLEGE.

*Reprinted from* THE LANCET, *August 15, 1908.*

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GENTLEMEN,—Among the problems of ophthalmic surgery the solution of which has given rise to much speculation is that of the cause of the enophthalmos which follows an injury to the eye or orbit. Many hypotheses have been advanced to account for this apparently anomalous condition; so many, indeed, that they include a wide range of possibilities—possibilities, however, which are of interest mainly because of their variety. It is essential for our purpose to attempt some classification of these cases, and in doing so we may divide them under three heads: first, those in which there has been shrinking of the eyeball in consequence of fracture of the bony wall of the orbit; secondly, those in which the eyeball has sunk owing to the cicatricial contraction of the orbital connective tissue resulting from inflammatory processes; and thirdly, those in which the eyeball sinks after a blow upon the eyeball itself or over the region of the orbit more or less quickly without any apparent lesion. With regard to the first class, it is obvious that the cases are really displacement of the eyeball—that is, partial or complete dislocation of the globe following the injury to the orbital wall. Langenbeck, for example, describes a case in which the eye was displaced into the antrum of Highmore. Again, Schapringer narrates a case recorded by Smetius where a missing eyeball was located in the nasal passages. In the second class of cases the enophthalmos condition can also be readily understood seeing that the cicatricial contraction of the orbital connective tissue drags the globe backward and anchors it there. With regard, however, to the third class of cases the matter is a very different one. Here presumably we have to deal with a manifest condition for which no adequate explanation seems to be apparent. As

my experience has been limited to this latter class of cases so my remarks will be confined to the discussion of the facts and conclusions set forth in the literature of the subject, in addition to which I will draw attention to some data hitherto unnoticed which are especially interesting and no less important in this regard.

Some years ago it was stated that judging from the number of cases on record traumatic enophthalmos was of very rare occurrence. But while at that time the number recorded did not exceed 26, since then the list has been very considerably increased; so much so, that we may assume that the small number of cases above alluded to was due, not to the fact that this ocular anomaly had not been met with, but rather that its occurrence had been overlooked as a subject for clinical observation and report. Conceivably, therefore, if every ophthalmic surgeon were to recall and publish his experience in this regard we should probably find that traumatic enophthalmos was by no means a rare condition.

First it must be remembered that there is no such thing as a typical case of traumatic enophthalmos. Practically in all cases the signs are different. Nevertheless there are certain constant changes which may be enumerated as follows: (1) shrinking of the globe; (2) more or less ptosis; (3) narrowing of the palpebral fissure; and (4) diminution of tension. In respect to the ptosis, while it is possible that this is due to paralysis of Müller's muscle, the innervation of which is derived from the sympathetic, it is also possible that the drooping of the upper lid depends upon the loss of support which follows shrinking of the globe, and this would also account for the narrowing of the palpebral fissure. These signs, therefore, may be regarded as in some sense mechanical, and as such they are easy of explanation. On the other hand, the difficulty begins when we come to consider the cause of the enophthalmos and the change which leads to the diminution of tension.

I have said that practically, apart from these constant signs, each case of traumatic enophthalmos differs. The cases vary in the period of their onset, in their duration, and in their intensity. Let us consider what recorded cases have shown in respect to these various points. There is, for example, no rule so far as the period of onset is concerned. In some cases the globe begins to shrink at once; in others the shrinkage only begins to become apparent after some days, following the traumatism. Again, no rule exists as to the period of duration;

the enophthalmos may be only temporary, lasting no longer than 48 hours, as occurred in one of my own cases. On the other hand, the globe may remain permanently in an enophthalmos condition. Next, the greatest variety is displayed in the character and intensity of the symptoms. Even the shrinkage of the globe varies in degree. Thus in a case recorded by Purtscher, in which a man was kicked by a horse over the right frontal and temporal region, the right eye was noticed almost at once to have shrunken, and a year afterwards the shrinkage had advanced to such a degree as to enable an artificial eye to be worn over the globe, and yet the notes state that the shrunken globe was normal in appearance, that the movements in all directions were natural, that there was no deviation, and that nevertheless blindness was present owing to complete atrophy of the optic nerve. The atrophic condition of the disc was probably due to some direct retro-ocular injury which the nerve had received. But no mention is made of whether or not the cornea retained its sensibility, and in the circumstances it is certainly remarkable, assuming that it was not anæsthetic, how such an intensely sensitive structure as the cornea could have tolerated in contact with its surface the artificial eye which was used.

Again, the pupil may be the seat of almost any functional change which it is possible for it to manifest. It may, for example, be contracted, dilated, responsive or non-responsive to light, and fail or not, as the case may be, to react to drugs. In one of my own cases there was contraction, but the pupil showed no change either under the influence of light or atropine.

We next have to consider the paralytic effects noticed in the external ocular muscles—a somewhat frequent concomitant of these cases of traumatic enophthalmos. These muscular changes must be regarded rather as having only an adventitious relationship to the purely enophthalmos signs than as being directly associated with them. They indicate a more extensive lesion than that which is sufficient to cause the enophthalmos alone. Again, they may or may not be permanent. No rule exists as to the particular muscles which may be involved: there may be one, or two, or even more, and differences in degree of the loss of power are generally exhibited by each of those concerned. Sometimes the paralysis is complete, say, of the external rectus, while paresis only is shown of the inferior oblique. Or, again, the inferior rectus may be paralysed and the external and superior recti may exhibit only slight loss of power. In short,

almost any combination of paralyses or pareses may be present in the external ocular muscles in association with traumatic shrinkage of the globe.

We have now to consider the question of the vision. No rule exists as to how far the vision may or may not be affected in these cases. Enophthalmos, *per se*, has no disturbing effect upon the sight. On the other hand, when loss of vision is met with ample cause for this is to be found in some serious ocular lesion attributable to the primary traumatism. Thus the vision may vary between the normal and that of complete blindness, as in Purtscher's case, according as the ocular structures have or have not suffered from the effects of the injury. And yet it is not without interest to point out that while in one case a contusion of an eye may be followed by enophthalmos, without any discoverable lesion, a similar contusion may result in, say, a rupture of the choroid, in association with which no enophthalmos occurs. This dissimilarity in the effects in each case appears to introduce a paradox. Why is it, one may ask, that in one case enophthalmos results without any further lesion of the eye, while in the other the injury causes a rupture of the choroid without any signs of shrinkage of the globe? This is a point which in the present state of our knowledge cannot be absolutely determined. So far no opportunity has occurred of establishing post mortem the real nature of the lesion present in cases of traumatic enophthalmos. In the circumstances, therefore, we can only fall back upon speculation in the endeavour to solve the problem, a procedure upon which, as it would be scarcely profitable, I do not propose to embark.

Let me now discuss some of the theories which have been advanced to account for this condition of traumatic enophthalmos. The first to which attention may be directed and to which most notice has been drawn is that favoured by Beer, who maintains that the shrinkage of the globe is due to absorption of the orbital cellular tissue and fat caused by lesion of the trophic peripheral nerves. The difficulty in the acceptance of this theory is, in fact, the point that it fails to be sufficiently explanatory. If a trophic lesion was extensive enough to cause so marked a nutritional change as that leading to absorption of the orbital fat, it is difficult to understand how the eye could fail to avoid being involved in the process. Again, as the theory stands, there is nothing in pathology by which it could be supported. Moreover, as I shall show later, evidence can be produced from surgical practice by which it seems to be entirely dis-

proved. Van Duyse holds an almost similar view to that of Beer. He affirms that the shrinkage of the globe may be due to post-traumatic tropho-neurotic affections causing a gradual resorption of the orbital fat. Of course, this theory is impossible in view of the cases in which enophthalmos quickly follows the receipt of the traumatism. Schapringer, on the other hand, claims that the symptoms are due to paralysis of the sympathetic, and that as the result of the injury the sympathetic has been "bruised or haemorrhagically compressed." He cites in support of this view one of his cases in which the symptoms only persisted for three days. It is, however, noticeable that apart from the paralysis of Müller's muscle no theory is advanced to explain how lesion of the sympathetic could cause enophthalmos.

Thus from these expressions of opinion it will be gathered that the belief obtains to the effect that enophthalmos follows some disturbance of the ocular innervation, some failure of the nerve-supply to the eye and orbit, some arrest of nutrition of the orbital, if not ocular, structures. And here it may be conceived, in the absence of any post-mortem evidence verifying or otherwise the theories advanced, that further discussion upon the subject would become profitably impossible. With this assumption up to a short time ago I would have felt bound to agree. While in the course of preparing this lecture, however, I happened to recall the series of cases published by Mr. Jonathan Hutchinson, jun., recording his results of the treatment of facial neuralgia by partial or complete removal of the Gasserian ganglion. Before, however, proceeding to discuss in detail the facts which his cases disclose, let me briefly revert to a description of the sources of innervation of the eye and orbit. There are, of course, the optic nerve and the ophthalmic division of the fifth nerve which is altogether sensory in function; then there are the motor nerves—namely, the third, fourth, and sixth; and lastly the sympathetic branches which are derived partly from the carotid but mostly from the cavernous plexus. With the recollection of the whole innervation of the eye and orbit before us, it is easier to speculate respecting the particular nerve-supply which is presumably involved in the cases in which enophthalmos occurs. But while neither pathology nor physiology has enabled light to be thrown upon this question it so happens that surgery has come to our aid.

I will now refer to the series of cases, those of Mr. Jonathan Hutchinson, jun., in which by the removal, wholly or partially, of the Gasserian ganglion for facial neuralgia the

innervation of the eye and orbit was almost entirely arrested. And yet despite this fact Mr. Hutchinson informs me that throughout his cases, now numbering 26, no enophthalmos was noticed. The inference from this is obvious. In the light of the opinions which have been expressed regarding the close connexion which is believed to exist between loss of innervation of the eye and orbit and traumatic shrinking of the globe, it might reasonably be assumed that the whole or partial removal of the Gasserian ganglion could scarcely fail to be followed by enophthalmos, and yet, as the cases above alluded to show, practically two-thirds of the innervation of the eye may be arrested without this result ensuing. Thus we are led to infer that the nutrition of the eye, in the sense indicated, is not interfered with when two-thirds of its nerve-supply are cut off. This naturally leads us to inquire more closely into the relations and influence of the remaining third—namely, that of the sympathetic branches supplied from the cavernous plexus. It would be well at this juncture briefly to refer to the distribution within the orbit of these particular branches. They are three in number—namely, one which joins the third nerve, a second which accompanies the fourth nerve, and a third which follows the ophthalmic division of the fifth and is continued forward to the lenticular ganglion either in association with, or distinct from, the nasal nerve. Now a word, also, as to the lenticular ganglion itself. It forms, so to speak, the soul of the eye within the orbit. From it are derived the sensory, motor, and sympathetic nerve-supply to the eyeball—the sensory from the nasal branch of the ophthalmic division of the fifth, the motor from the branch of the oculo-motor to the inferior oblique muscle, and lastly, as we have seen, the sympathetic from the cavernous plexus. It is obvious that these anatomical details have a very important bearing upon the probable cause of the enophthalmos with which we are especially dealing—that is to say, the inference is clear, in cases of such an apparent failure of nutrition of an eye, that our first inquiry should necessarily be directed to the nerve centre upon which the integrity of that function depends. The nerve centre in question is undeniably the lenticular ganglion, seeing that it controls both the nutrition of the eyeball as well as innervates the involuntary muscular fibres of the eye and the upper lid through its sympathetic root.

Let us now change somewhat the venue of our discussion and proceed to inquire into the symptoms of those cases in which some injury has been received to the cervical sympathetic. Lesion of the cervical sympathetic, of course,

implies paralysis of the sympathetic fibres distributed to the eyeball, the effects of which Horner was the first fully to describe. The symptoms include: (1) partial ptosis, owing to paralysis of Müller's muscle, to which reference has already been made; (2) narrowing of the palpebral fissure; (3) contraction of the pupil, owing to loss of innervation of the dilator fibres; (4) diminution of intra-ocular tension; (5) sinking back of the eyeball; and (6) changes in the vascular supply of the face on the affected side. My attention was first directed to these symptoms some years ago when I was asked to see a young woman in whom they were well marked, from whose neck there had recently been removed a mass of enlarged tuberculous glands. In the course of the operation, no doubt, as sometimes happens, the cervical sympathetic had incurred some injury, from which the paralytic signs had ensued. It remains now to compare these signs with those for the most part observed in cases of traumatic enophthalmos. It will be seen at once that they are almost exactly parallel. Variations, of course, in degree and extent of the paralytic effects will naturally occur, and these will have to be taken into account in making the comparison. Nevertheless, so far as the four cardinal and constant signs are concerned of traumatic enophthalmos—namely, shrinkage of the globe, partial ptosis, narrowing of the palpebral fissure, and diminution of tension, the coincidence with the symptoms of paralysis of the ocular sympathetic is obvious and undeniable.

Having now, so to speak, localised the cause of the signs observed in traumatic enophthalmos we have still to determine that hitherto difficult problem of why shrinkage of the globe should occur as the result of the loss of sympathetic innervation. I have said hitherto difficult problem advisedly, inasmuch as in the light of the facts already referred to, the solution thereof appears to be easy. To complete our inquiry, however, it is necessary to turn to the description of Tenon's capsule; thus we find, quoting from "Quain," that "from the capsule of Tenon septa of fascial connective tissue spread outwards mainly in the direction of the recti muscles, all of which receive delicate investments from the structure: a layer also spreads between the muscles and the periosteal lining of the orbit. In two places these septa of fascia are of greater strength than elsewhere—viz., on the inside towards the lacrymal sac and eyelids, and on the outside towards the margin of the orbit, where it is united with the external ligament of the palpebræ and periosteum. In several parts of these fascial structures,

more especially in those last mentioned, plain or unstriped muscular fibres have been detected, and these, together with the elastic connective tissue, are conceived to act in restoring the position of parts after the cessation of the action of the voluntary muscles. These involuntary muscles in the septa are under the influence of the cervical sympathetic nerves." The septa in question are known as the cheek ligaments; the function which they discharge, sufficiently explained in the quotation given, is plainly an important one, otherwise they would scarcely have been provided with involuntary muscular tissue. To sum up, the loss of innervation to the check ligaments allows the eyeball to recede, and the cause of the sympathetic paralysis is probably due to concussion, contusion, or some lesion of the ocular nerve centre in the orbit —namely, the lenticular ganglion, whose sympathetic root is involved in the injury. In short, traumatic enophthalmos depends, not upon trophic nerve changes leading to absorption of the fat and connective tissue of the orbit, but upon disturbance of the sympathetic innervation of the eyeball, probably commencing in the lenticular ganglion. Maklakoff has observed that of the ocular muscles affected in these cases the external rectus most frequently shows signs of paralysis, and in accounting for this he advances the theory that probably some extravasation of blood takes place from the ophthalmic artery, the abducens, owing to its proximity to the artery, being thus particularly liable to be involved. But clearly hæmorrhage into the orbit, such as this theory implies, would tend to cause, not shrinkage of the globe, but exophthalmos, and, moreover, in the circumstances of so gross a change, we should expect to find evidence of optic neuritis due to compression of the optic nerve at its entrance into the orbit. On the other hand, attention has recently been drawn to the fact that the external rectus derives its nerve supply, not only from the sixth nerve, but also from some fibres distributed to it from the sympathetic, a statement which closely accords with the view which attributes the cause of these cases of traumatic enophthalmos to disturbance of the sympathetic innervation and would in some measure also account for the frequency with which the external rectus is involved. It might be asked, not without interest, "How can sympathetic paralysis be present in those cases in which the pupil exhibits a normal reaction?" The obvious reply is that the paralytic signs present are dependent upon the extent of the injury which the sympathetic has received and the special fibres which have been involved. It is scarcely logical to expect that the paralytic signs should

be the same in every case. Again, as showing the limitation of the lesion we do not find that paralysis of the ocular sympathetic is followed by vaso-motor disturbance of the skin of the corresponding side of the face, whereas such a change is always observed accompanying lesion of the cervical sympathetic.

Three cases of the kind of traumatic enophthalmos under discussion have come under my observation, to the notes of which I may briefly refer.

Case 1.—A male, aged 39 years, was admitted under my care into the West London Hospital with the following history. He was struck over the right eye and orbit by the handle of a jack which knocked him down, causing loss of consciousness. On admission two hours afterwards there was found laceration of the upper and lower lids, requiring the use of sutures. No complaint was made of the eye itself, which was apparently uninjured. After nine days treatment in the ward he was discharged to attend as an out-patient. A week later it was seen that marked enophthalmos of the right eye was present. He also complained of diplopia, which on examination was found to be due to paresis of the right internal rectus; the pupil was slightly larger than that in the left eye but reacted sluggishly to light. Accommodation was unaffected. R. V. $\frac{6}{18} + 0 \cdot 75$ cyl.: axis V. $= \frac{6}{6}$; L. V. $\frac{6}{6}$. Four weeks later the enophthalmos was the same, but he stated that the diplopia had almost gone. The fundus was normal. T. —.

Case 2.—A boy, aged 12 years, was admitted into the West London Hospital with the history of having been forcibly struck over the right eye by the handle of an umbrella. There was much ecchymosis of both lids. In the course of ten days the blood was absorbed and then it was seen that enophthalmos was present. Movements of the globe were free in all directions; the pupil was contracted and reacted to light. Subsequently it responded well to atropine. T. —; fundus natural. R. V. $\frac{6}{9}$; L. V. $\frac{6}{6}$. Some months later the signs had undergone no change, the enophthalmos being just as manifest.

Case 3.—This case was seen in private. The history was as follows. A young lad, aged 16 years, was shot in the right eye by a fellow schoolboy with an air gun. On examination some hours afterwards, previously to which vomiting had occurred several times, I found an abrasion of

the upper and lower lids, the lead pellet having struck the eye in a glancing direction—the gun was fired from an upper storey window while the patient was advancing towards it in the playground below. The conjunctiva and cornea were intact and no evidence existed of penetration of the globe. T. normal. There was, however, much photophobia. On the following morning the globe was a good deal injected; the photophobia was less and the eye felt fairly comfortable. Shortly after my visit on the previous evening it was stated that another attack of vomiting had occurred, but some sleep had been obtained during the night. T. normal; pupil contracted. The next day chemosis was observed on the inner side; partial ptosis; T. —. Pupil only slightly dilated despite guttæ atropæ: obvious enophthalmos. Within 48 hours, however, these signs had entirely passed off. No enophthalmos was apparent, the pupil was widely dilated, the eyeball was nearly pale, and the tension was normal. Subsequently the vision was examined and $\frac{6}{6}$ was barely reached. The fundus showed choroidal disturbance in the macular region. No actual rupture of the choroid was visible, but numerous fine guttate pigmentary changes were noticeable. In a few days later the vitreous contained many opacites. After a month the vitreous became nearly clear and the vision improved to $\frac{6}{24}$.

The above three cases are illustrative of the variety which cases of traumatic enophthalmos may present. In the first case, besides the signs of sympathetic paralysis, there was paresis of the external rectus, but while, when the patient was last seen, this paresis appeared to be passing off, the enophthalmos signs had undergone no improvement and have probably proved to be permanent. In the second case, on the other hand, the enophthalmos was unaccompanied by any collateral or associated sign; the lesion was clearly confined to the sympathetic from which no improvement is to be expected. The third case is interesting as showing temporary sympathetic paresis; such uncomplicated cases of traumatic enophthalmos of brief duration are rare, as is shown by the literature of the subject.